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PERSISTENCE OF IMPAIRED HEAT TOLERANCE FROM ARTIFICALLY INDUCED--ETC(U)
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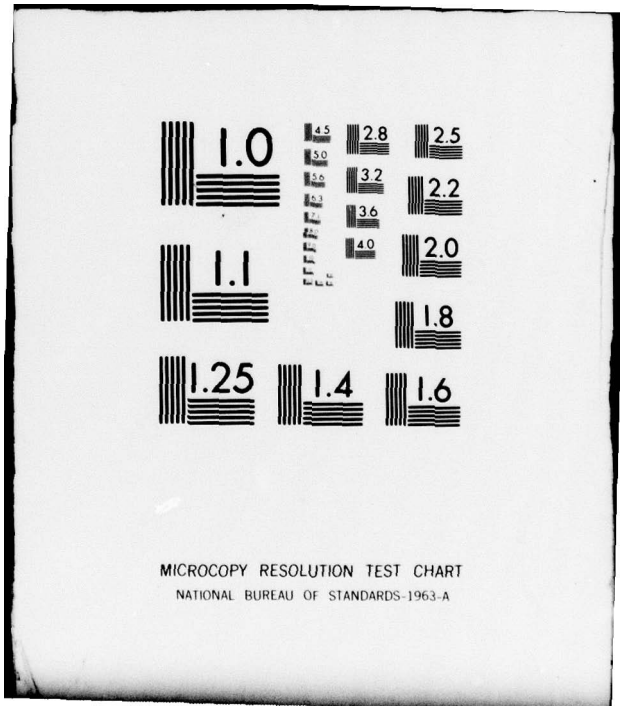
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Ten volunteer subjects were heat acclimatized to 48.9°C (T_a), 20% rh for 7 days to complete a 100 min walk on a level treadmill (1.56 m s^{-1}). Subjects were then divided into experimental (6 Ss) and control (4 Ss) groups; both groups had comparable means for the measured variables rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), heart rate (HR) and sweat rate. Miliaria rubra (heat rash) was then induced on the experimental subjects by wrapping them for 3 days in polyethylene plastic. All 6 developed marked miliaria with involvement of 4(

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70% of the total body surface area. The other 4 subjects served as a control group for the normal decay of heat acclimatization. All subjects were re-exposed to walking in the heat on the 7th day after unwrapping, by which time rash was clinically undetectable, and again 14 days after unwrapping. On the first test (day 7) only one of the rashed group, and on the second test (day 14) only two could complete the 100-min walk; the control group finished without difficulty on boty days. Body heat storage for the rash group was nearly three times that of the control group on day 7 and twice as great on day 14; measurements of mean body temperature ($\bar{T}_b$) on the rash group indicated a much greater heat stress when compared to their own pre-rash acclimatized values or those of the control group. These data demonstrate the potential of "healed" miliaria in the etiology of clinical heat illness.

PERSISTENCE OF IMPAIRED HEAT TOLERANCE FROM ARTIFICIALLY INDUCED MILIARIA RUBRA

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Running head: IMPAIRED HEAT TOLERANCE AND HEAT RASH

Ten volunteer subjects were heat acclimatized to 48.9°C (T_a), 20% rh for 7 days to complete a 100 min walk on a level treadmill (1.56 m s^{-1}). Subjects were then divided into experimental (6 Ss) and control (4 Ss) groups; both groups had comparable means for the measured variables rectal temperature (T_{re}), mean skin temperature ($\bar{T}_{sk}$), heart rate (HR) and sweat rate. Miliaria rubra (heat rash) was then induced on the experimental subjects by wrapping them for 3 days in polyethylene plastic. All 6 developed marked miliaria with involvement of 40 to 70% of the total body surface area. The other 4 subjects served as a control group for the normal decay of heat acclimatization. All subjects were re-exposed to walking in the heat on the 7th day after unwrapping, by which time rash was clinically undetectable, and again 14 days after unwrapping. On the first test (day 7) only one of the rashed group, and on the second test (day 14) only two could complete the 100-min walk; the control group finished without difficulty on both days. Body heat storage for the rash group was nearly three times that of the control group on day 7 and twice as great on day 14; measurements of mean body temperature ($\bar{T}_b$) on the rash group indicated a much greater heat stress when compared to their own pre-rash acclimatized values or those of the control group. These data demonstrate the potential of "healed" miliaria in the etiology of clinical heat illness.

Index terms: heat rash; performance time; exercise; core temperature; skin temperature; mean body temperature; heart rate; sweat rate; body heat storage; heat acclimatization; anhidrosis

Miliaria rubra (heat rash) has long been a disease associated with infants and with those persons required to work under conditions of very high environmental temperatures. Prior to World War II, little emphasis was placed upon miliaria rubra and its physiological and medical importance, other than as a nuisance entity. During World War II, a heat illness, variously described as thermogenic anhidrosis (20), tropical anhidrotic asthenia (1,19), and heat exhaustion II (5) was first recognized. Typically, the patient with tropical anhidrotic asthenia (the now commonly accepted terminology) displays complaints of gradual to rapidly progressing inability to tolerate physical activity in the heat with an associated newly recognized inability to sweat from all body surfaces from the neck down. A history of preceding, widespread miliaria rubra is usually present, although in some cases it may have preceded the heat intolerance by several weeks and a history of miliaria is not obtained. In some cases the work-heat intolerance is so severe that the simple activity of taking a few steps is rapidly associated with symptoms of extreme weakness, malaise, headaches, anorexia, and visual disturbances.

The initial reports of this syndrome (5,20) recognized the striking absence of sweating and the associated miliaria rubra. However, the authors emphasized possible central or peripheral neurological damage or possible exhaustion of the eccrine sweat glands as the etiology. It was subsequently established that the etiological process involved the primary occlusion of the epidermal sweat duct unit of the eccrine sweat gland with subsequent inability to deliver sweat to the skin surface (1,17). Although classically, persons with tropical anhidrotic asthenia required several months to recover, a number of individuals, reported as having tropical anhidrotic asthenia, recovered in 2 to 3 weeks (1,5,20). These individuals apparently had either ordinary miliaria rubra, or as described in a few of these

patients, no history or clinical evidence of any form of miliaria (1,20).

No subsequent studies on the possible relationship between ordinary miliaria and subsequent anhidrosis were available until 1967 with the resurgence possibly stimulated by the Vietnam conflict. Prior to this time the study of this entity depended solely on the presence of clinical cases. In 1967, Sulzberger et al. (16) demonstrated that a simple occlusive wrapping of a volunteer's skin with polyethelene film for a period of 48 hours was followed by the development of typical miliaria rubra lesions. Utilizing this method, Griffin et al. (3) demonstrated that the duration of hypohidrosis following one bout of laboratory-induced miliaria rubra was 14 to 21 days and was directly related to the severity of the induced rash. A most significant observation was the presence of hypohidrosis in the absence of detectable clinical skin disease, as the induced miliaria in these volunteers appeared to have completely resolved by 7 days after the induction period. Thus, the skin of these men appeared clinically normal despite the presence of marked occlusive disease of their eccrine sweat glands. In those patients reported as having tropical anhidrotic asthenia without history or clinical evidence of miliaria (1,20), it is certainly possible that they could have had a preceding, mild bout of miliaria which had recently resolved (two to three weeks) and been forgotten only to have left a significant residual hypohidrosis. Nevertheless, no studies to our knowledge have been conducted to investigate the physiological responses to work-heat tolerance associated with ordinary miliaria.

The following study was undertaken to determine if one bout of ordinary miliaria rubra (prickly heat) could result in a significant reduction in man's ability to function physiologically under heat and work stress. Also, the possible changes in functional ability to deal with heat and work stress up to 14 days after the induction of the miliaria were evaluated.

METHODS

Ten healthy, Caucasion male volunteers were utilized in the study. All subjects were fully informed with regard to experimental risk and gave their written informed consent. These men had no history of preceding heat rash or other skin diseases except perhaps mild facial acne vulgaris. Initially, the men were heat acclimatized by walking (1.56 m s^{-1}) on a level treadmill for 100 min in a controlled environmental chamber at a temperature (T_a) of 48.9°C and a relative humidity of 20% (27.8°C, T_{wb}) with a wind velocity of 1.4 m s^{-1}. These volunteers performed this exercise daily for 7 days, when all could satisfactorily complete the walk without evidence of any continuing rise in core temperature or of physical exhaustion. Subjects wore shorts, socks and boots during these chamber exposures. Experimental variables routinely evaluated during all chamber trials were mean body temperature, heat storage, heart rate, sweat rate and tolerance time. Sweating was evaluated on selected areas of the body surface of each subject by the method of Tashiro et al. (18). In this method, dry bromphenol blue is dissolved in pure acetone in a 5% solution; this solution is then mixed in silicone (Dow-Corning Corporation) stopcock grease in a ratio of 1:1 in volume. The mixture is carefully compounded to assure uniform distribution of the bromphenol blue particles and is stirred continuously until the acetone completely evaporates. The application of 10 coats of this material to the skin allows testing of selected areas of the body for the presence or absence of clinical sweating. As sweat occurs, it is trapped in the silicone grease, and the water in the sweat reacts with the dye turning it blue. The degree of sweating can be estimated, using a five point scale, as absent (5), very minimal (4), minimal (3), moderate (2) or heavy (1).

After heat acclimatization, the ten volunteers were carefully matched

according to heart rate, rectal and skin temperatures, and performance time with six selected as test subjects and four as controls. The experimental subjects (n=6) had an average age (mean ± SE) of 21.3 ± 1.4 yr; height, 172.1 ±1.4 cm; weight (nude), 72.3 ± 2.2 kg, and body surface area of 1.85 ± 0.03 m^2 while the control subjects (n=4) had an average age (mean ± SE) of 22.0 ± 0.8 yr; height, 173.1 ± 1.6 cm; weight (nude), 71.5 ± 2.7 kg, and body surface area of 1.85 ± 0.04 m^2. Miliaria rubra was then induced over 70% of the body surface of the 6 experimental subjects by wrapping the entire body, except for the head, neck, hands, feet, and perineum, in polyethylene film (Saran Wrap^R, Dow-Corning Chemicals, Midland, Michigan). The film was reinforced with elastic plastic tape (Blendum^R Tape, Minnesota Mining and Manufacturing Company, St. Paul, Minnesota) and held in place by elastic ballet tights. The method was a modification of that used by Sulzberger et al. (16). The men remained wrapped for a period of 72 hours. The control subjects wore elastic ballet tights for 24 hours and then were allowed to wear ordinary street clothing. Just prior to the removal of the tights and wraps, all subjects were exposed to heat in the environmental chamber at 48.9°C (20% rh) for 30 min to maximize the induced miliaria. Following removal of the wrapping, photographs were taken and the men were observed to determine the degree and distribution of the induced rash. The volunteers were then allowed to bathe and to resume their usual daily activities with the exception of heavy exercise and exposure to sun.

Seven days after the removal of the wrapping, the bromphenol blue sweat test material was applied to selected sites of all men, both experimental subjects and controls. The men then were subjected to a repetition of the treadmill walking and heat stress test under conditions identical to the 7-day acclimatization period.

This treadmill test was repeated again 14 days after the removal of the wraps.

During all environmental chamber experiments, skin temperatures were monitored with a three-point thermocouple skin harness (chest, calf and forearm) and mean weighted skin temperature ($\bar{T}_{sk}$) calculated according to Burton (2). The rectal temperature (T_{re}) was recorded from a thermistor probe inserted ~ 10 cm. Mean body temperature ($\bar{T}_b$) was calculated as 1/3 $\bar{T}_{sk}$ plus 2/3 T_{re}. Heart rate (HR) was measured by palpation during rest and after each 25 min of walking. Subjects were given 250 ml of water during the 10-min rest period prior to the chamber exposure and were encouraged to drink the same amount at 4 points during the exposure; a total consumption of about 1.25 L. Total body weight losses were determined from pre- and post-walk measurements on a gram scale for calculation of sweat rate without correction for respiratory losses. Body heat storage was calculated according to Burton (2).

Statistical Analysis.

A mixed factorial analysis of variance design (7) was used with each subject receiving all combinations of the factors (day and time), but the subjects separated into groups (control and rashed). This was utilized for both the acclimatization phase and the periods following the rashing. Experimental variables evaluated were mean body temperature, heat storage, heart rate, sweat rate and tolerance time. When significance was found, critical differences were calculated (6) to determine where the mean differences existed.

RESULTS

After heat acclimatization, experimental (rashed) and control groups were formulated with subjects matched for a variety of physiological responses. The experimental and control groups had average (mean $\pm$ SE) values for final rectal

temperature (T_{re}) and final heart rate (HR) of 38.7 $\pm$ 0.16°C and 135 $\pm$ 8 bts/min (experimental) and 38.5 $\pm$ 0.23°C and 133 $\pm$ 5 bts/min (control) which illustrates the physiological similarity between groups before the induction of experimental rash. Strict interpretation of the statistical findings between groups after rashing may be somewhat misleading. Many of the statistical analyses were conducted from data only when both groups were experimentally intact (mean body temperature, heat storage, heart rate, sweat rate). Thus, the effect of voluntary and/or early experimental termination of many of the rashed subjects due to impaired performance must be remembered.

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The mean tolerance time of all subjects for the 1st day of heat acclimatization (D_1) of 85.5 $\pm$ 4.4 min was significantly lower ($P < 0.025$) than the 7th (D_7) day mean of 100 min, but there was no significant effect ($P > 0.05$) due to groups (experimental = 85.7 $\pm$ 7.0 min, 100 min; control = 85.2 $\pm$ 5.2 min, 100 min). Figure 1 compares the average tolerance time between the experimental and control groups during the final day of heat acclimatization (D_7) with the responses from the two test days (7 and 14 days after unwrapping for the experimental rashed group). The mean tolerance time for the rashed group after 7 days of unwrapping was significantly lower ($P < 0.05$) than the control group on that day and was also significantly lower than its own group mean for the 7th heat acclimatization day. Although the average performance time was still reduced by 20 min on the 14th day after unwrapping for the experimental group, this difference was not statistically significant compared to the controls ($cd_{.05}$ = 28.3 min).

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There was no significant effect due to groups (experimental and control) during heat acclimatization for mean body temperature ($\bar{T}_b$). However, there was a significant day by time effect ($P < 0.05$) with means at each 10-min time period from 0 to 60 min differing between days (day 1 and day 7). There were also significant differences ($P < 0.05$) among the time means within each day. Figure 2 compares $\bar{T}_b$ between the experimental and control groups on the 7th day of heat acclimatization contrasted to responses 7 days after unwrapping of the experimental rash group, or 14 days after unwrapping. Although no differences were exhibited between groups during the last day of heat acclimatization, the group differences comparing responses 7 days after unwrapping of the rashed group with the last acclimatization day are marked and are significant ($P < 0.05$) at 20, 30, 40 and 50 min. Significant group differences after 14 days of unwrapping were not found until 50 min. The experimental rash group had the higher $\bar{T}_b$ at these time periods. It should be remembered that all control subjects completed the 100-min experimental period while many rashed subjects voluntarily terminated exposure after 50 min.

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The analysis of group heat storage comparisons utilized the difference between initial and final heat storage values (Δ heat storage in kcal) for calculating purposes. No significant group differences were found for body heat

storage comparisons between the 1st and last heat acclimatization days. However, there was a significant day by time interaction ($P < 0.01$) with the day 7 (last day) mean significantly lower than the day 1 mean at each corresponding 10-min interval. Figure 3 presents the comparison of body heat storage values between the experimental and control groups on either the last heat acclimatization day (day 7), the last day of acclimatization contrasted to the responses 7 days after unwrapping of the rashed group, or 14 days after unwrapping. The statistical differences between groups for body heat storage are the same as for $\bar{T}_b$. Again, the marked differences between groups from 20-50 min of exposure after 7 days of unwrapping should be noted.

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Sweat rate analyses (kg/hr) showed no significant differences for either acclimatization or during testing (last acclimatization day compared to 7 or 14 days after unwrapping rashed group). The sweat rates were calculated on a per-hour basis, but due to early removal of some subjects on most days, the rate was lower than it might have been, had all subjects completed the full 100 min. Nevertheless, Figure 4 shows that for all group comparisons the control group had higher sweat rates but these differences were not significantly greater. Utilizing the method of Tashiro et al. (18), observations of clinical sweating in the rashed areas compared to the same areas in the control subjects were made immediately following the rash inducement exposure to heat (following 72 hours of being wrapped). When the 6 rashed subjects were evaluated, the average severity of sweating was about midway between "moderate" and "minimal" (2.57 ± 0.13). This

compared with noted "heavy" (1.00) sweating in all areas on all four controls with one local exception (one subject had only "moderate" sweating on right upper arm).

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Heart rate analysis showed no significant effect due to groups. The initial heat acclimatization day mean differed from the final day mean ($P < 0.05$) and the mean at each time (0, 25, 50 min) differed from the other two ($P < 0.05$). As illustrated in Figure 5, in the analysis of heart rate responses during the last acclimatization day compared to 7 or 14 days after unwrapping, the combined groups show only a significant time effect ($P < 0.01$) with the means at 25 and 50 min being significantly higher than 0-time. However, it should be noted that group comparisons after 7 or 14 days of unwrapping show a progressively greater difference in heart rate responses with time; the rashed group has the higher value but the difference does not reach statistical significance.

DISCUSSION

The results of this study, although involving small numbers of volunteers, were dramatic. Extensive experimentally induced miliaria over 40 to 70% of the body surface (mean, 56.8 $\pm$ 4.9%) of 6 healthy male volunteers led to a significant and serious impairment of their ability to perform work under conditions of high environmental temperature. This impairment was present both 7 days and 14 days after the 72-hour miliaria induction period had been completed. Seven days after the induction of the miliaria, two subjects in the experimental group developed heat exhaustion ($T_{re} \geq 39.5^{\circ}C$ and/or HR $\geq$ 180 bts/ min) in the first 50 min of exposure, two more subjects by 75 min, and a fifth subject by 90 min of testing.

Thus, only one of the six experimental rash subjects was able to complete the 100-min walk seven days after the induction of miliaria, and he had a final T_{re} of 39.4°C and was at the point of collapse. Even fourteen days after the induction of the heat rash, only two out of the six members of this test group were able to complete the 100-min walk; the other four again experienced heat exhaustion. All of these men had been heat acclimatized prior to the miliaria induction period, and could easily perform the heat and work stress tests prior to the induction period. Some loss of heat acclimatization occurred in the control group but all satisfactorily completed the 100-min walk on both occasions (7 and 14 days after experimental rash induction). Previous observations on similar test subjects (male soldiers) showed physiological loss of heat acclimatization to be minimal up to 18 days of non-exposure to heat (9).

Average performance time of the experimental rash group was reduced by 32.3 min and 20.7 min after 7 and 14 days of unwrapping, respectively. Concomitant and significant physiological differences were seen between the rash and control groups. Where intact groups could be compared statistically, mean body temperature ($1/3\ \bar{T}_{sk} + 2/3\ T_{re}$) after 50 min of exposure was 0.9°C and 0.4°C higher for the rash group than control group values 7 and 14 days after unwrapping, respectively. Body heat storage for the rash group was nearly three times that of the control group on day 7 and twice as great on day 14 ($P < 0.05$). The degree of heat intolerance and hyperthermia seen in these experimental subjects compares with reported responses of individuals with anhidrotic ectodermal dysplasia which is an hereditary disorder characterized by aplasia of the eccrine sweat glands (19).

Significant to this study was the observation that, except for one subject who developed moderate folliculitis, no difference could be detected between the skin

of the rashed subjects and that of the control subjects at the time of the 7-day and 14-day heat-work stress tests. Only after the initiation of the heat tests, was marked eccrine sweat gland occlusion apparent, as manifested by absence of clinical sweating in the previous rashed areas of the experimental subjects. Thus, these experiments clearly showed that extensive, experimentally-induced miliaria rubra can lead to a serious decrement in man's ability to withstand heat and work stress even after the clinical miliaria has resolved.

The incidence of prickly heat is very high for individuals living in tropical areas or working in any hot environment. It is also very common in infants who are over-clothed by apprehensive parents. Individual susceptibility varies but some state that essentially all are candidates for this disease. Sulzberger and Emik (15) reported that 66% of several hundred men studied on Guam had some miliaria rubra. The general term anhidrosis may be defined as the inability of the body to produce and/or deliver sweat to the skin surface (12). More specifically, miliaria is the generic term for a group of disorders of the skin due to sweating in the presence of an obstruction of the sweat duct (14). Most specifically, miliaria rubra is associated with a physical plugging of the affected sweat glands (14). However, the most important factor would appear to be the number of sweat glands implicated by the antecedent attack of miliaria (8). Although a number of studies have been published on the clinical observations of miliaria rubra (1,3,5,15,16,20), no studies have been reported on the actual physiological performance decrements associated with heat rash. By blockage of the sweat duct, miliaria produces anhidrosis and thereby diminishes heat tolerance (8). Since man loses heat from his body by means of evaporation, anhidrosis denotes the lack of a crucial physiological defence against thermal stress (13).

Because it lowers heat tolerance, miliaria probably predisposes an individual towards anhidrotic heat exhaustion. The sequence of events may well be as follows: miliaria, loss of function in those sweat glands with keratotic plugs, compensatory hyperactivity of unaffected sweat glands, fatigue and failure of the sweat mechanism, and anhidrotic heat exhaustion (8). By causing a rise in the chloride concentration of sweat, miliaria and its sequelae may, under specified conditions, predispose toward classic heat exhaustion. Horne and Mole (4) state that "prickly heat is commonly assumed to be merely a local affection of the skin, but the evidence suggests that, like some other cutaneous syndromes, it may also be a manifestation on the surface of some general metabolic disturbance". This disturbance appears to primarily involve the pituitary-adrenal system (12). Certainly, more than closure of the gland pores seems to be involved. The observations from this study of a lower sweat rate (kg/hr), while not significantly lower, support the possibility of fatigue or failure of the sweat glands as a major contributing factor in the reduced performance of individuals with heat rash under work-heat stress. The noted "minimal to moderate" sweating of the heat rashed group compared to "heavy" sweating response of the control group supports this conclusion. The key to these conclusions is the unraveling of the nature of sweat-gland fatigue within the context of functional decrements in performance due to heat rash.

Acclimatization to heat was conducted in these subjects to provide a reasonably equivalent experimental baseline from which to conduct the study. Heat acclimatization has been found by some to result in a reduction in sweat chloride provoked primarily by a salt deficit ~~(10,11)~~, while the chloride concentration is found to be greatly increased in the sweat from areas involving miliaria.

(8,12). These incongruous findings may point out the over importance placed on increased chloride concentration in the pathophysiology associated with miliaria particularly as it affects performance. Nevertheless, since acclimatized individuals are better able to maintain fluid balance than those not acclimatized, the importance of the reduced tolerance times of these experimental subjects is further magnified.

The findings from this investigation clearly illustrate that one severe bout of laboratory-induced miliaria rubra results in a marked reduction in performance and an increase in several physiological responses of these individuals compared to matched controls. Had the humidity been elevated to that of jungle conditions, it is quite possible that the results would have been even more dramatic. The distribution of sweat glands differs in various areas of the body. The effect of heat rash on different areas of the body and associated work-heat performance responses is not known.

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This research was performed by Drs. Robert J. T. Joy, Tommy B. Griffin and Ralph F. Goldman, and presented by them at the 1968 International Physiological Congress, Washington, D.C.

The views, opinions, and/or findings contained in this report are those of the author(s) and should not be construed as an official Department of the Army position, policy, or decision, unless so designated by other official documentation. Human subjects participated in these studies after giving their free and informed voluntary consent.

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FIGURE LEGENDS

FIG. 1. Differences in tolerance time between experimental rash and control groups while walking (1.56 m s^{-1}) in the heat (48.9°C, 20% rh) for an attempted 100 min. Comparisons are made between the last (7th) day of heat acclimatization, and seven or fourteen days after unwrapping the experimental group.

FIG. 2. Comparison between experimental rash and control groups for mean body temperature ($\bar{T}_b$) on the final day of acclimatization to heat, seven days post rash, and fourteen days post rash.

FIG. 3. Comparison between experimental rash and control groups for the difference between initial and final body heat storage values (Δ heat storage in kcal) on the final day of heat acclimatization, seven days post rash, and fourteen days post rash.

FIG. 4. Differences in sweat rate (kg/hr) between experimental rash and control groups while walking (1.56 m s^{-1}) in the heat (48.9°C, 20% rh) for an attempted 100 min. Comparisons are conducted between the last (7th) day of heat acclimatization, and seven or fourteen days after unwrapping the experimental group.

FIG. 5. Comparison between experimental rash and control groups for heart rate (bts/min) on the final day of heat acclimatization, seven days post rash, and fourteen days post rash.

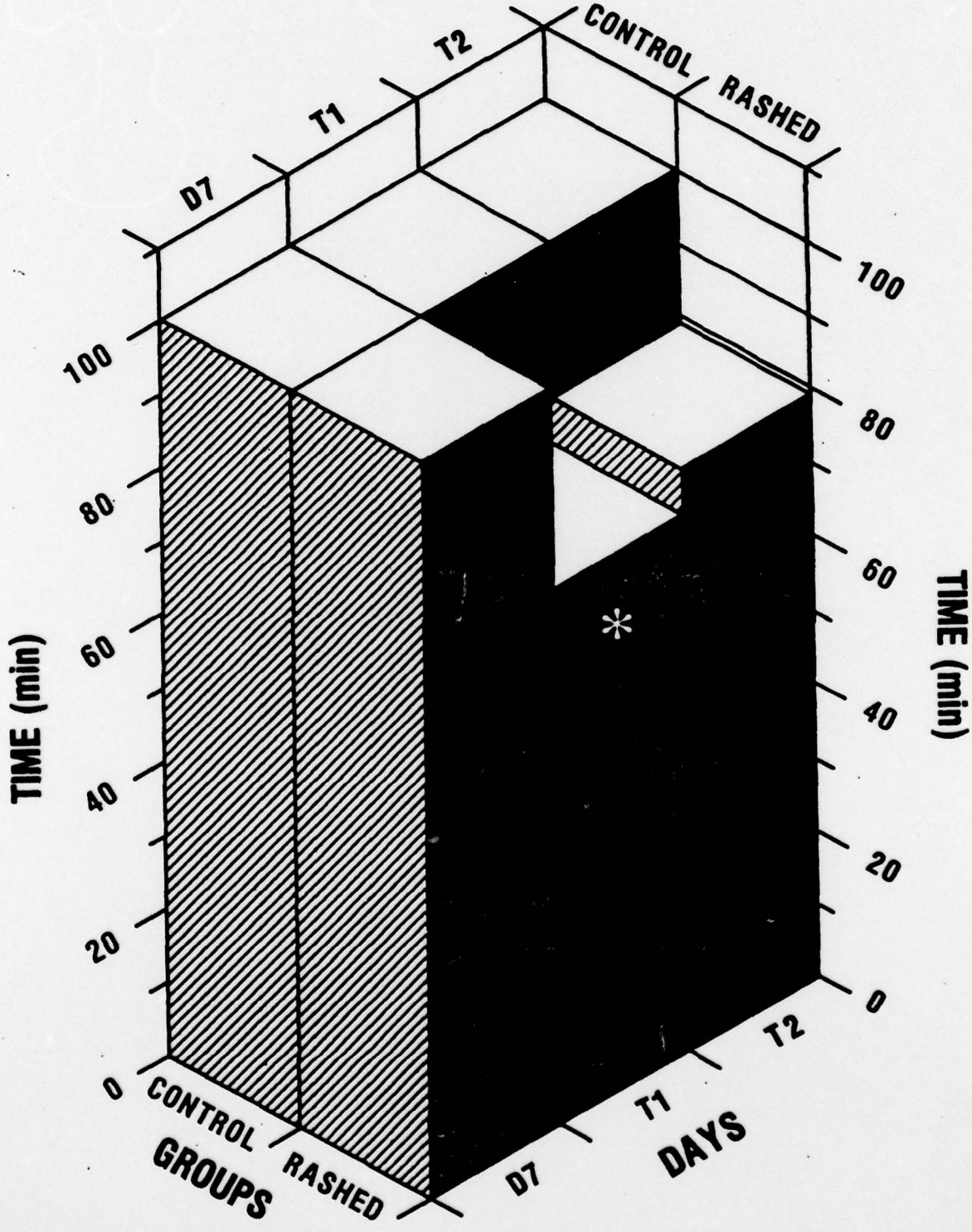

CONTROL
RASHED
T2
T1
D7
100
80
60
40
20
0
TIME (min)
CONTROL
RASHED
GROUPS
D7
T1
T2
DAYS
*

FIG. 1

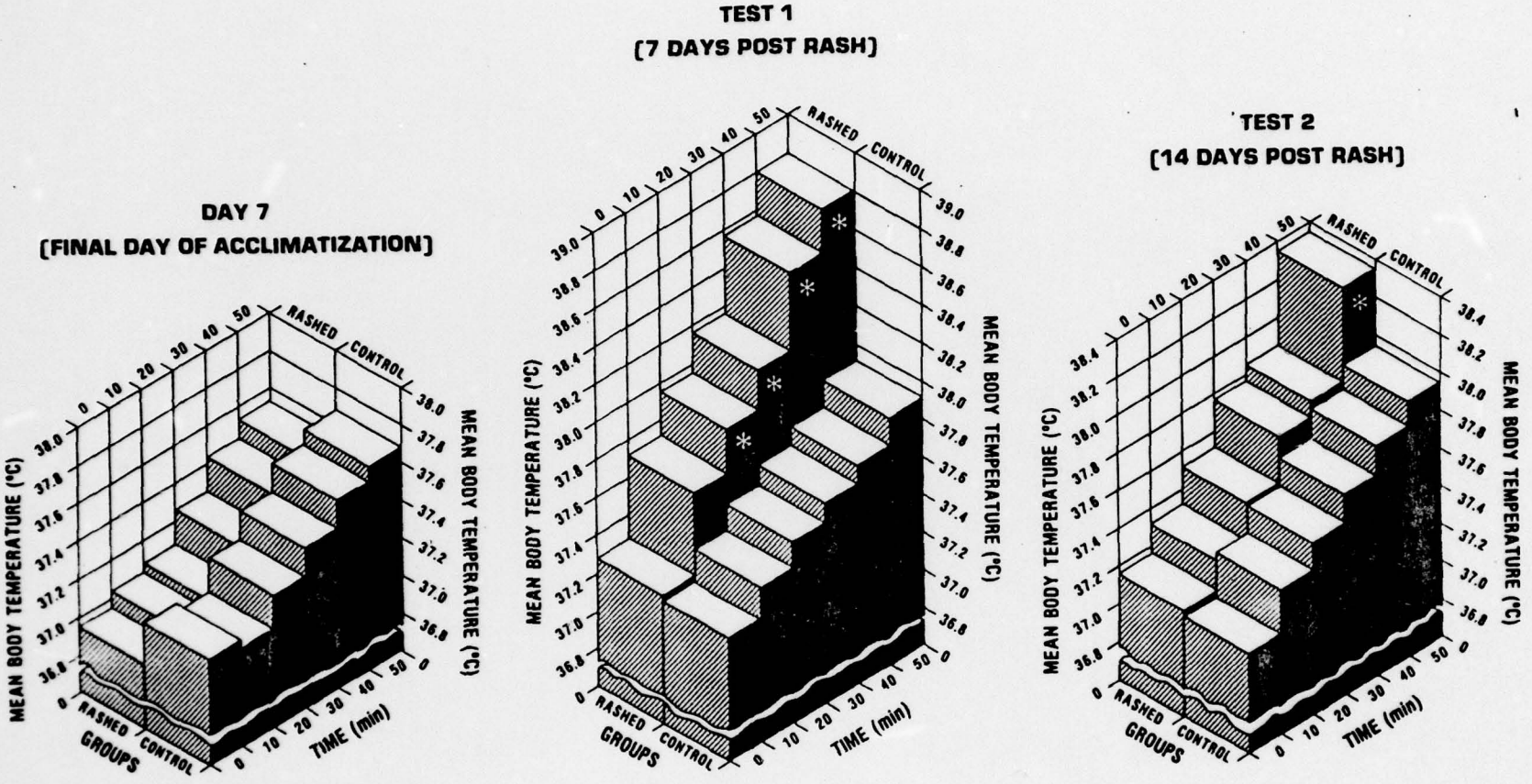
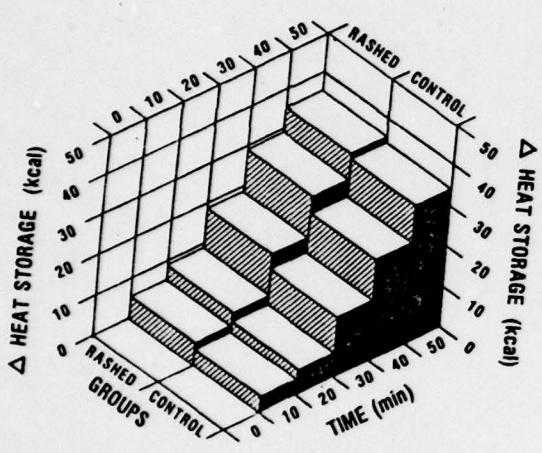
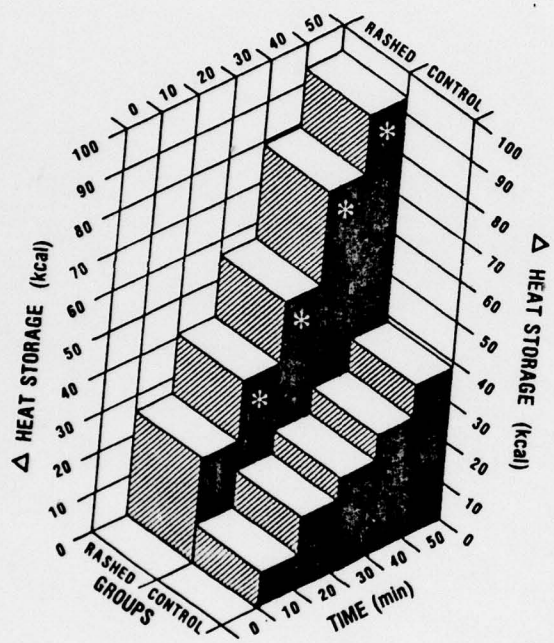

DAY 7
(FINAL DAY OF ACCLIMATIZATION)
TEST 1
(7 DAYS POST RASH)
TEST 2
(14 DAYS POST RASH)
MEAN BODY TEMPERATURE (°C)
MEAN BODY TEMPERATURE (°C)
RASHED
CONTROL
GROUPS
TIME (min)
0
10
20
30
40
50
36.8
37.0
37.2
37.4
37.6
37.8
38.0
38.2
38.4
38.6
38.8
39.0
*

FIG. 2

DAY 7
[FINAL DAY OF ACCLIMATIZATION]



TEST 1
[7 DAYS POST RASH]



TEST 2
[14 DAYS POST RASH]

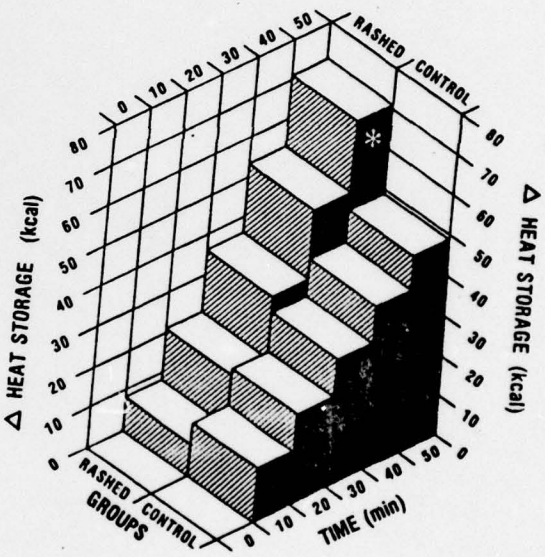


Fig. 3

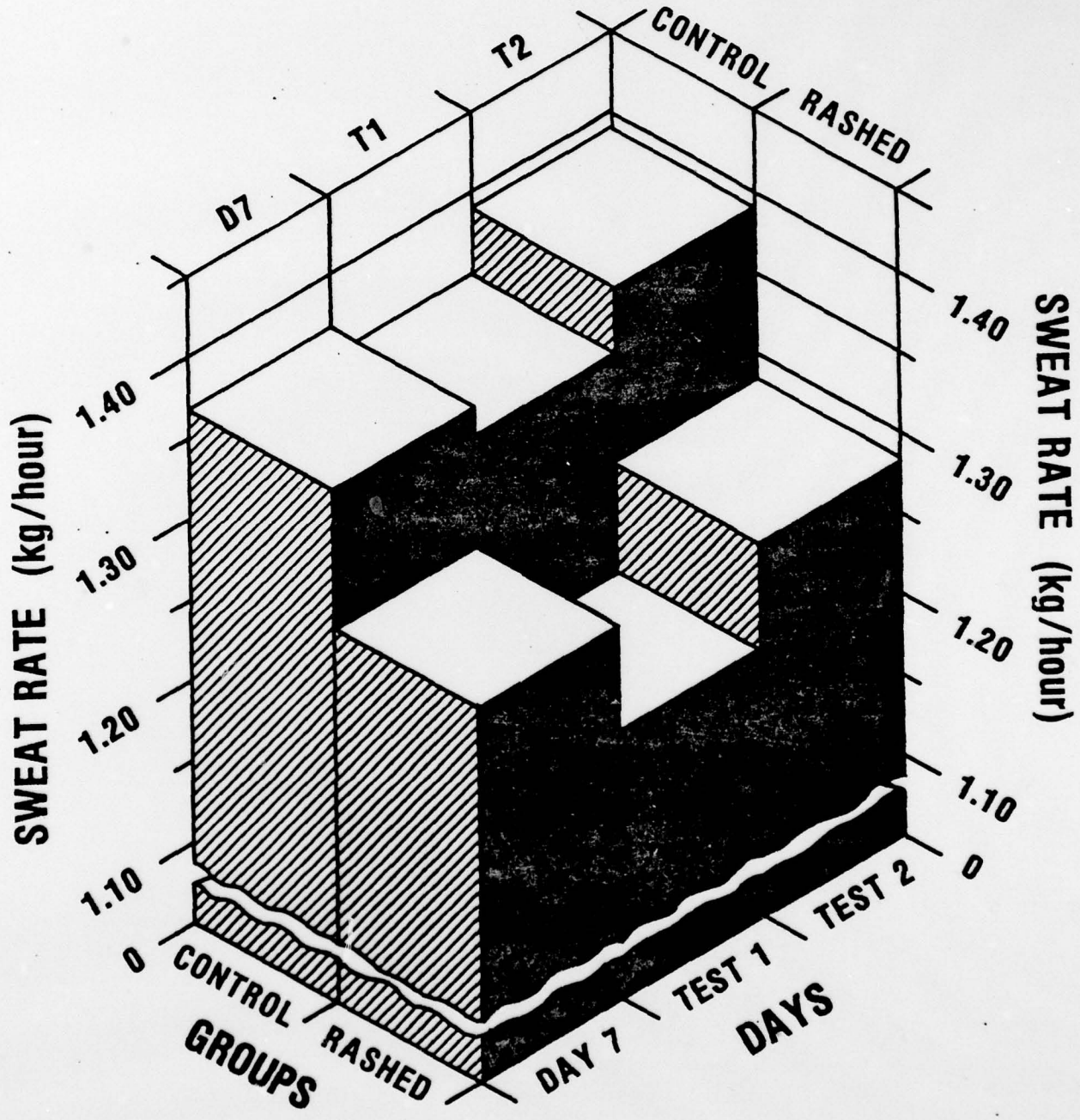
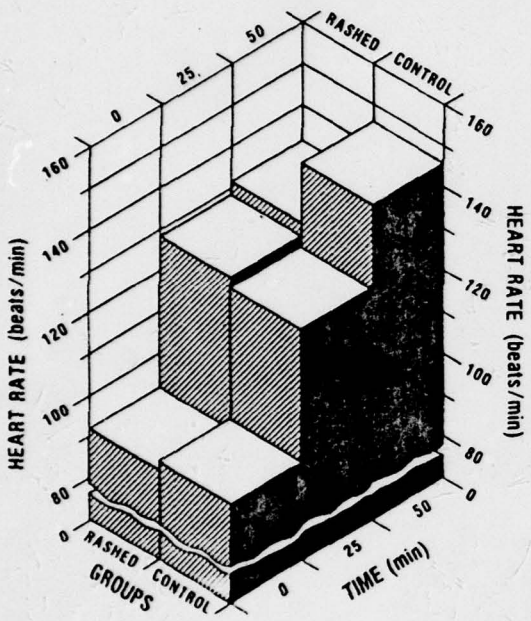
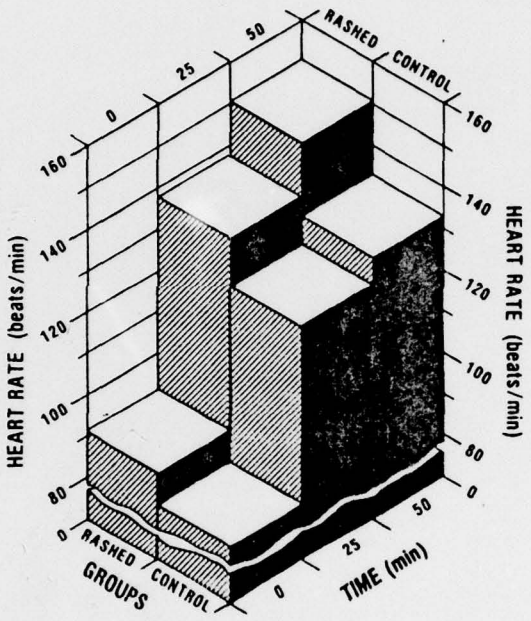

SWEAT RATE (kg/hour)
1.40
1.30
1.20
1.10
0
CONTROL
RASHED
GROUPS
DAY 7
TEST 1
TEST 2
DAYS
D7
T1
T2
CONTROL
RASHED
SWEAT RATE (kg/hour)
1.40
1.30
1.20
1.10
0

FIG. 4

DAY 7
[FINAL DAY OF ACCLIMATIZATION]



TEST 1
[7 DAYS POST RASH]



TEST 2
[14 DAYS POST RASH]

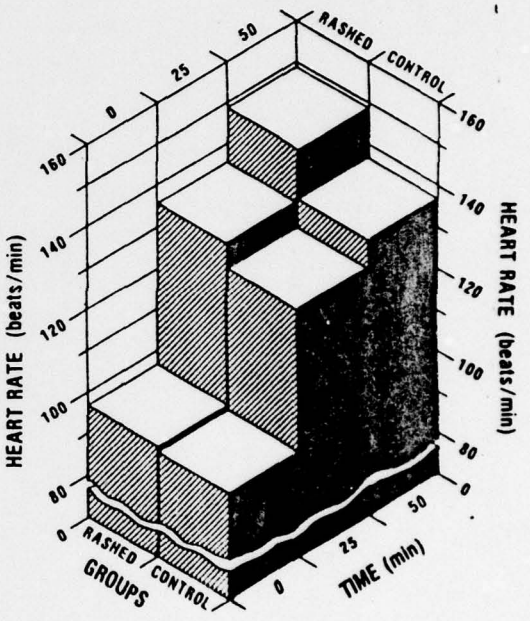


FIG. 5